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Abstract
In this paper, a simple general equilibrium model *à la* Solow is developed to capture the impact of AIDS on economic growth. To this end, a benchmark model due to Cuddington and Hancock (1994) is extended in various directions. In particular, the sharply declining life expectancy patterns are clearly re°ected in the enlarged model through a generic Ben-Porath mechanism. AIDS-related health expenditures are incorporated as well. Using up-do-date optimal forecasting methods, the model applied to South Africa shows that while a relatively short term assessment might not reveal any dramatic AIDS growth e®ect, the medium/long run impact can be truly devastating. In particular, the heavy trends in mortality and life expectancy currently induced by AIDS are shown to be potentially at least twice more detrimen-tal for per capita economic growth in the period 2020-2030 compared to 2000-2010.

Key words: Epidemics, life expectancy, economic Growth, AIDS
JEL codes: C61, C62, O41

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1 Introduction

The impact of epidemics on the economic development process has witnessed a tremendous revival recently due to the worldwide AIDS pandemic and its persistence. Specially the case of sub-Saharan Africa is the focus of an increasing number of empirical studies in this respect. Actually, the interest of economists has not been exclusively restricted to AIDS. Many studies have also been devoted to the Black Death or Spanish flu epidemics. In particular, the empirical evidence on the economic effects of the Spanish flu is highly disputed. The question turns out to be whether say the Spanish flu did induce a fall or rise in per capita income growth in the short run. Empirically, the question is very far from trivial. First of all, one should recall that the Spanish flu occurred after the first world war, and therefore, part of the phenomena that took place in our period of interest is also probably due to the post-war adjustment dynamics. Second, and more generally, disentangling the effects of the epidemic is markedly complicated because one has to control for many other potential explanatory factors, like urbanization, the sectoral composition of the economy, initial GDP and other numerous variables. This seems like a daunting task. However, some very careful studies on this issue have already come out. Brainerd and Siegler (2003) and Bloom and Mahal (1997a) are among the very best[1].
Bloom and Mahal studied the case of the Spanish flu in India. Precisely, they looked at the acreage sown in India across 13 Indian provinces. They found no relationship between the magnitude of population decline and the variation in acreage sown per capital across provinces. In a more detailed investigation, Brainerd and Siegler focused on the impact of the Spanish flu in the US. In their study, the dependent variable is the growth rate of per capita income from 1919-1921 to 1930, and the primary explanatory variable is the number of flu and pneumonia deaths per 1000 persons in each state of the US reported in 1918 and 1919. A nice feature of this study is the inclusion of many more variables to control for other potentially important factors (like education levels, initial income, agricultural share of personal income, ethnic composition...etc...). The main result of their econometric regressions is the significant and positive impact of the Spanish flu on the growth rate of per capita income: "...the flu coefficient ranges between 0.219 and 0.235...and is always statistically significant at the 5 percent level or lower..." (Brainerd and Siegler, 2003, page 17). And the authors conclude that along with conditional convergence and the rise of education, the Spanish flu does significantly matter in the economic growth history of US.

Just like the Spanish flux epidemic's empirical literature, the debate is strikingly disputed among economists concerning the economic consequences of AIDS, specially in

[1]Another related known and even classical investigation on the growth effect of epidemics is due to Jack Hirshleifer in his celebrated book, *Economic Behaviour in Adversity*, 1987. Hirshleifer examined the case of the plague which killed one-third of the European population between 1348 and 1351. He suggested that the plague by sharply reducing the population size led to a rapid and persistent rise in real wages for laboring classes.

sub-Saharan Africa countries. In a highly influential paper, Bloom and Mahal (1997b) found no significant effect of AIDS on the growth rate of per capita income, and no evidence of reverse causality. They used simple cross-section regression models on a sample of 51 countries from 1982 to 1992. The estimated coefficients were found typically small and insignificant.

Such result went at odds with the results obtained at the same time on more theoretically founded models *à la* Cuddington and Hancock, which generally predicted a relatively important growth impact of AIDS. An important reference here is the paper of Over (1992). The author used a relatively sophisticated computable general equilibrium model with three classes of workers, and rural versus urban production. This ultimately allowed him to study the impact of several AIDS scenarios, based on different assumptions about relative levels of HIV infection in educated Vs uneducated workers...etc...In the most reasonable scenarios (according to Over), the effect in the 10 most affected sub-Saharan Africa countries would be 0.6 percentage point over the period 1990-2025 if all treatment costs were financed from savings.

More recently, some new contributions putting forward the human capital channel have found large effects of AIDS on growth. For example, Corrigan, Glomm and Mendez (2005) have used a calibrated OLG model to analyze particularly the effect of the drop in life expectancy on investment, and the large generation of orphans produced by AIDS. Their results are completely in line with Cuddington, Hancock and Over. McDonald and Roberts (2005) use an econometric model combining growth and health capital equations. Applied on African countries, the model predicts substantial effects of the epidemic: the marginal impact on income per capita of a one percent increase in HIV prevalence rate is minus 0.59%. The authors conclude that while the human and social costs of the HIV/AIDS epidemic are major causes for concern, their results do indicate that the macroeconomic effects of the epidemic are by no way negligible. Finally Ferreira and Pessoa (2003) find that in the face of an AIDS-like epidemic schooling time can decline by half, which cannot be neutral for long-run economic growth.

At the same time, another controversy emerges as to the effect of AIDS on fertility. Whether the epidemic has decreased or increased the fertility rates in the countries most affected by AIDS/HIV is of course a sensitive point if one aims at gathering a clear diagnosis on the impact of the epidemic on per capita variables, which happens to be the most frequently used indicators of welfare. In a highly controversial paper, Young (2005) claims that AIDS should sharply lower fertility for two main reasons. On one hand, the epidemic has certainly reduced the willingness to engage in unprotected sexual activity. On the other, the high mortality of adult males and the resulting increasing scarcity of labor are likely to increase the value of woman's time. Both channels are possibly strong enough to induce a durable decrease in fertility, which may cause future consumption per capita to rise. Using a Barro-Becker based empirical model, the author finds that in the case of South-Africa, this decreasing fertility engine is so strong that it dominates the

human capital channel put forward by Corrigan, Glomm and Mendez (2005) for example. Hence, AIDS might well be interpreted as a "gift of the dying" for future South African generations.
Nonetheless, such a finding has been challenged by some authors, including the very devastating paper by Kalemli-Ozcan (2006) who studied the fertility issue on a panel of 44 African countries over the period 1985-2000. She finds that the HIV/AIDS epidemic affects the total fertility rates positively and the school enrollment rates negatively[2]. As it can be inferred from the previous statements, there does not seem to be any consensus on the fertility channel. We shall adopt here the position taken by most demographers (like those of the US Census Bureau), that it is the low perception of HIV/AIDS risks in sub-Saharan Africa makes quite unrealistic the drastic changes in fertility behavior postulated in some economic models. This lack of awareness, also pointed out by UNAIDS in its successive annual reports, is likely to reduce the impact of the fertility channel.
However, a compelling evidence on the sharp negative effect on schooling has been gathered over the two last decades, and we argue that this is a key point for getting through the empirical literature on AIDS impact on economic growth.

In this paper, we shall extend the accounting framework of Cuddington and Hancock in order to account for the human capital channel, an aspect left in the dark by the authors. We shall also amend the model in order to cope with some of the important criticisms raised by Bloom and Mahal (1997b) in their posterior econometric study. We shall develop these amendments in detail in the next section. Our aim is to develop a simple general equilibrium framework *à la* Solow, which in addition to the typical factor accumulation mechanisms inherent to the neoclassical growth theory, also encompasses some of the salient characteristics of AIDS, notably the effect on schooling time and/or life expectancy. Incidently, we will show in our application to the South African case that there might not be any fundamental conflict between the results obtained by authors like Bloom and Mahal (1997b), and others like Corrigan, Glomm and Mendez (2005): A relatively short term assessment *à la* Bloom and Mahal might not reveal any dramatic AIDS growth effect, while a long term perspective, relying on the evolution of key demographic variables like mortality and life expectancy, might yield just the contrary. The paper uses some up-to-date forecasting techniques. The forecasting procedure starts with the estimation of the parameters of an assumed model, that is the assumed underlying data generating process. Gaussian $ARMA(p,q)$ processes are assumed for this purpose. We report in detail the crucial elements of the estimation and the forecasting procedures associated with such processes.

The paper is organized as follows. Section 2 presents the benchmark Cuddington-Hancock set-up, and lists the criticisms raised against the model and method used by these two authors. Section 3 develops an enlarged model incorporating in particular the

[2]This outcome can be rationalized in a model with precautionary demand for children in face of uncertainty about child survival.

schooling and life expectancy trends. This model is then applied to the South African case in Section 3. After a thorough description of the econometric methodology implemented, we give a summary of our findings.

2 A simple accounting set-up: Cuddington and Hancock (1994)

Cuddington and Hancock have proposed a very simple accounting exercise to measure to which extent AIDS harms (or could have harmed) GDP growth in some sub-Saharan Africa countries. In their 1994 *Journal of Development Economics* paper, they have applied it to Malawi. Cuddington and Hancock's set-up is a very useful tool to get an immediate idea about the extent of damages caused by AIDS. We shall develop it in the next section, and then give the arguments raised against it.

2.1 The benchmark model

The set-up is of the Solow type, and it is based on the production function:

$$Y(t) = A(t)\ K(t)^{\theta}\ \left([1 - \phi(t)\ x(t)]\ \mu(t)\ L(t)\right)^{1-\theta}.$$

$Y(t)$ is total output (or GDP) of the economy. It is produced with a Cobb-Douglas technology with two factors, physical capital, $K(t)$, and labor, $L(t)$, θ being the capital share. With respect to the standard Solow model, some epidemic-specific variables are introduced. In particular, a new term, μ_t, appears, supposed to capture labor-specific productivity, and in this sense, it could be interpreted as a human capital indicator. Additionally, the new production function includes a term $x(t)$, which reflects the role of morbidity: a larger $x(t)$ amounts to a lower effective labor effort due to morbidity. In a more general setting, such a loss may be reduced by medical care. In Cuddington and Hancock, it is assumed exogenous: the authors consider all the situations when $x(t)$ ranges from 0 to 1. Finally, the production function includes a term $\phi(t)$, which stands for the AIDS prevalence rate at date t. Of course, the loss in productivity due to morbidity depends on the spread of the epidemic, and such a characteristic is reasonably captured by the prevalence rate.

Another highly interesting aspect of the framework developed by Cuddington and Hancock is the introduction of the age structure of the labor force in the production function. Of course the epidemic does not affect in the same way all the age classes, such a refinement is therefore not only useful, it is necessary. Call $E(t)$ the effective labor force, that is

$$E(t) = [1 - \phi(t)\ x(t)]\ \mu(t)\ L(t),$$

such a magnitude may be rewritten in a straightforward way taking into account the age distribution:

$$E(t) = \sum_i \left[1 - \phi_i(t)\; x_i(t)\right]\; \mu_i(t)\; L_i(t), \tag{1}$$

where i stands for the age index. Cuddington and Hancock have made further simplifications. First, they assume $x_i(t) = \xi$, where ξ is a constant comprised between 0 and 1, $\forall i$ and $\forall t$. Second, they work with $\mu_i(t) = \mu_i$, $\forall t$. Third, they even postulate a simple quadratic form for age-specific productivity:

$$\mu_i = \rho_1 + \rho_2\; (i - \bar{i}) - \rho_3\; (i - \bar{i})^2, \tag{2}$$

where ρ_k, $k = 1, 2, 3$ are three positive numbers, and where $\bar{i}$ is, for example, the minimal age to enter the labor market[3]. Equation (2) merely states that productivity is a quadratic function of age, and in this sense, it exclusively captures the experience determinant of productivity.
The set-up is completed by a capital accumulation equation:

$$K(t) = s\; Y(t) + (1 - \delta)\; K_{t-1}, \tag{3}$$

where s stands for the saving rate of the economy (assumed constant by Cuddington and Hancock), and with K_0 given. To be more precise, the authors retrieve a term $x\; H_t$ from the right hand side of equation (3), where H_t stands for total health expenditures, and x is the fraction of these expenditures related to AIDS. We shall consider here the saving rate s in a broader sense, it is the fraction of income not spent in consumption and health expenditures, which allows us to write capital accumulation as usual.

Using the demographic projections issued by the World Bank for Malawi, Cuddington and Hancock have studied to which extent the AIDS epidemic has affected and will affect GDP and GDP per capita in this country in the period 1985-2010. The World Bank projections entailing a comparison in terms of mortality and morbidity between AIDS and without AIDS configurations, Cuddington and Hancock have also conducted this comparison in more economic terms using such a valuable demographic information (ie. in variables L_{it}, and A_{it}). Indeed, they distinguished between an extreme AIDS scenario and a medium one: In the former scenario, average real GDP growth over the period 1985-2010 would cost 1.2 to 1.5 percentage points relative to the non-AIDS counter-factual case, while in the latter the figure drops to only 0.2 to 0.3[4]. Concerning annual growth rate of real GDP per capita, the study shows an average depression of 0.25 percentage points through the year 2010.

[3]Cuddington and Hancock took $\bar{i} = 15$.

[4]The range is generated by letting ξ and x move from 0 to 1.

2.2 Criticisms

Whatever the defects of the framework used, which does not incorporate (and is not aimed at incorporating) all the economic mechanisms induced by an AIDS-lie epidemic, the results of Cuddington and Hancock have the merit to suggest that the growth effects of AIDS could be indeed sizeable in some African countries. As we have mentioned before, some subsequent papers have however raised some serious reservations against the framework used, specially Bloom and Mahal (1997b). We shall summarize the criticisms in a few points.

1. Bloom and Mahal questioned the treatment of Cuddington and Hancock on the specific ground of the labor market in sub-Saharan Africa countries: according to them, the presence of a labor surplus could mitigate the output losses that might otherwise be associated with AIDS morbidity and mortality. Apparently, such a criticism does not seem to have a decisive scope, at least in certain countries: In a companion paper, Cuddington (1993) has shown that his results are not that sensitive to the presence of surplus labor in the Tanzanian case.

2. A clearer shortcoming of the setting is the treatment of morbidity: the variable $x(t)$ is taken exogenous (and indeed, it is constant) while it should depend on medication. We shall amend this aspect of the original modelling closely.

3. Another criticism, raised by Bloom and Mahal, has to do with the way health expenditures enter the accumulation equation. In Cuddington and Hancock's setting, health expenditures show up exclusively as a decrease in savings channeled into capital accumulation. This might not be the case in real life: health expenditures are also detrimental to ordinary consumption expenditures. Overall, there is a very serious issue of how to deal with savings in a context of epidemics. In our enlarged model, we shall use the analysis and findings of Freire (2002) to overcome this important difficulty.

4. A last criticism would be the way productivity, through variable μ_i, is modelled, only relying on experience. Clearly, such a variable should also reflect the education level and other related socioeconomic determinants (like the gender). Also, the time-independence could be questioned in a more long term perspective: With life expectancy dropping from about 60 years to 40, one is tempted to suspect that human capital accumulation would be toughly affected, which would induce a downward trend in productivity. Actually, with the numerical values considered by Cuddington and Hancock for parameters ρ_i, $i = 1, 2, 3$ and $\bar{i}$, the productivity variable μ_i is maximal when the individual is 60 years old, which is roughly (and terribly) inconsistent with the recent evolution of life expectancy in sub-Saharan Africa.

We shall amend Cuddington-Hancock's set-up in the next section.

3 An application to AIDS in South Africa

3.1 Data

We now move to the empirical work. We start with a brief review of the data used for the main economic and demographic variables of the model and their source.

- *Economically active population Estimates and Projections, by age and sex (1980-2020).* The data comes from International Labor Organization.

  The database contains world, regional and country estimates and projections of the total population, the activity rates and the economically active population (labour force) by sex and five-year age groups (from 10 to 64 years and 65 years and over). These estimates and projections are for international comparisons and are neither superior nor necessarily inferior to national estimates and projections, which are produced using country-specific additional information. The economically active population comprises all persons of either sex who furnish the supply of labour for the production of goods and services during a specified time-reference period.

- *HIV-seroprevalence (percent, 1980-2015).* The data is provided by U.S. Census Bureau, International Programs Center.

  Estimated HIV adult prevalence trends from 1980 to 2015. These estimates were derived from the Epidemic Projection Package, an epidemiologically sound computer model that allows for a "best fit" of HIV prevalence data from antenatal clinic women who come in for their first antenatal visit. The HIV prevalence is defined as the percentage of women surveyed testing positive for HIV. Each year a national survey of HIV prevalence among women attending public antenatal clinics in South Africa is conducted by the Department of Health. The Annual HIV antenatal survey provides South Africa with annual HIV trends among pregnant women and further provides the basis for making other estimates and projections on HIV/AIDS trends.

- *HIV^+ (1990-2015).* The Demographic impact of HIV/AIDS in South Africa follows from National Indicators for 2004. This indicator is defined as the number of people infected.

- *Life expectancy at birth by age and sex (1920-2060).* The data is provided by World Development Indicators, Health Nutrition and population and League of Nations, Northwestern University. This is the number of years that a new born could live if the normal conditions of mortality at his birth should be the same ones throughout its life.

- *Total health expenditure (1960-2000).* We use Health Nutrition and Population database.

- *Percentage expenditure of AIDS in the total health expenditure (1960-2000).* The data is extracted from South African Budget Review, 2003/04 and Estimates of National Expenditure, 2003.

- *Unemployed by age and sex (2000-2003).* The data comes from International Labor Organization.

  The series on unemployment shown here relate in principle to the entire geographical area of a country. In 1982, the Thirteenth International Conference of Labour Statisticians adopted a new Resolution concerning Statistics of the Economically Active Population, Employment, Unemployment and Underemployment, in which the definition of unemployment is revised. The new definition is to a large extent similar to the earlier definition adopted by the Eighth Conference. It, however, introduces certain amplifications and modifications concerning, in particular, the criteria of seeking work and current availability for work, the statistical treatment of persons temporary laid off, persons currently available for work but not actively seeking work, etc. The changes are aimed to make it possible to measure unemployment more accurately and more meaningfully both in developed and developing countries.

- *GDP per capita (1960-2000).* The data comes from Penn World Table 6.1.

- *Saving rates with and without AIDS*. We use data from Freire (2002).

In what follows, we describe our enlarged Cuddington-Hancock model.

3.2 An enlarged Cuddington-Hancock accounting set-up

Our starting point is the following: as shown by many authors, the observed fall in life expectancy in most sub-Saharan Africa countries, one of the main consequences of the AIDS pandemic, should have induced a decline in schooling time, in addition to other dramatic effects on savings incentives. Ferreira and Pessoa (2003) find that in the face of an AIDS-like epidemic schooling time can decline by half, which is in our view quite fundamental for long run growth rate. In order to adapt the benchmark Cuddington-Hancock model to account for such crucial features, we introduce the following modifications.

1. Concerning the production function, we have incorporated AIDS-related health expenditures in the measurement of productivity, exactly as in Corrigan, Glomm and Mendez (2005)[5]. In order to capture the gender-specific characteristics of the epidemics, we also distinguish between males and females (index $f = 1, 2$):

[5]Since it is a Solow model, we don't have to determine the optimal pace of health expenditures given the demographic and epidemiological characteristics of AIDS. This is done properly in Feichtinger, Tsachev and Veliov (2005), and Almeder *et al.* (2006).

$$E(t) = \sum_{i,f} \left[1 - \phi_{i,f}(t)\ \Phi\left(m_{i,f}(t)\right)\right]\ \mu_{i,f}(t)\ L_{i,f}(t), \tag{4}$$

where $m_{i,f}(t)$ measure AIDS-related health expenditures for an individual belonging to the age class i and with gender f at time t. The introduction of a gender variable seems to us an important step. A crucial recent feature of HIV/AIDS in sub-Saharan Africa (and notably in South Africa) is the increasing percentage of females affected. In its 2004 report, UNAIDS mentioned that close to 60% of HIV/AIDS infected people are females in sub-Saharan Africa, the youngest being the most exposed to the infection risk for some obvious reasons.[6]

2. The productivity variables μ are specified in order to capture the effect of the declining life expectancy on productivity at work. We do this as follows. Notice that with the benchmark specification (2), the age at which μ_i is maximal is equal to $\bar{i} + 2\ \frac{\rho_2}{\rho_3}$. With a declining life expectancy, such a magnitude is likely to go down. This comes mainly through the well-known Ben-Porath mechanism, recently pointed out by Boucekkine, de la Croix and Licandro (2002), according to which a decreasing life expectancy lowers the schooling time. We simply capture this mechanism by endogenizing the coefficient ρ_2 and assuming the following ad-hoc functional relationship:

$$\rho_2(i, f, t) = \alpha_{i,f}\ \nu_{i,f,t}^{\beta},$$

where $\nu_{i,f,t}$ is the life expectancy at birth of workers of age i (thus born at $t - i$) and of gender f. $\alpha_{i,f}$ and β are calibrated[7] . With such a new specification, the age-specific productivities become time and gender dependent via life expectancy:

$$\mu_{i,f,t} = \rho_1 + \rho_2(i, f, t)\ (i - \bar{i}) - \rho_3\ (i - \bar{i})^2, \tag{5}$$

For the calculation of ρ_2, we set for the reference year, $\rho_2(i, f, t_0) = \alpha_i \nu_{i,f,t}^{\beta} = 0,005$ for a given β, then we calibrate $\alpha_i = \frac{0{,}005}{\nu_{i,f,t}^{\beta}}$, where 0 indicates the base year (2000). The determination of α_i at the basic year allows us to determine the ρ_2. We set the values of the other parameters as follows: $\rho_1 = 0,8$, $\rho_3 = -0,0001$ and we analyze the cases where β takes different values from 0 (no Ben-Porath mechanism) to 1 (a "linear" mechanism).

[6]See UNAIDS, UNFPA and UNIFEM, "Women and HIV/AIDS: Confronting the crisis", New-York and Geneva, 2004.

[7]In particular, the double sequence $\alpha_{i,f}$ is calibrated in such a way that $\rho_2(i, f, t_0) = \alpha_{i,f}\ \nu_{i,f,t_0}^{\beta}$, for β fixed, t_0 is the base year and $\rho_2(i, f, t_0)$ is equal to the constant coefficient ρ_2 considered by Cuddington and Hancock (1994).

3. *Determination of E, K and Y*

i) In the no AIDS case, the prevalence rates are set to zero. Then we have, $E(t) = \sum_{i,f} \ \mu_{i,f}(t) \ L_{i,f}(t)$ with $L_{i,f}(t) = (pans - u)h$, where *pans* indicates the active population without AIDS by age and sex, u is the number of unemployed people by age and sex , and h is the number of worked hours in a year (we use 49 weeks and 40 hours by week, that is $h = 1960$).

ii) As previously stated, our production function is of the type

$$Y_t = (1+\gamma)^t E_t^{1-\theta} K_t^{\theta},$$

then K_0 is given by the following relation

$$K_0 = K_{2000} = \left(\frac{Y_{2000}}{\gamma^0 E_{2000}^{1-\theta}} \right)^{\frac{1}{\theta}},$$

where Y_{2000} is the GDP of the base year (2000), and we set $\theta = 1/3$ and $\gamma = 0.012$. The physical capital $K(t)$ is obtained with the relation

$$K_{t+1} = s_t Y_t + (1-\delta) K_t,$$

where $\delta = 0.05$ (the values of the parameters δ, θ, γ are the same as in Corrigan et al., 2005).

iii) In the case with AIDS, we of course account for the AIDS prevalence rates $(\phi_{i,f}(t))$ and AIDS related expenditures, and we use life expectancy at birth with AIDS in the determination of $\mu_{i,f,t}$ as explained before. We also use the active population with AIDS in the determination of $L_{i,f}(t)$.

iv) However, we have faced a data unavailability problem when collecting AIDS-related health expenditures. Instead of the intended indicators $\Phi\left(m_{i,f}(t)\right)$, we have been forced to use $\Phi\left(m(t)\right)$ where $m(t)$ is given by the relation: $m(t) = txHIV \frac{M_t}{HIV^+}$ where $txHIV$ represents the percentage of AIDS related expenditures in the total health expenditures (M_t), and HIV^+ is the number of infected people. As in Corrigan et al. (2005), we pick the following analytical for $\Phi(.)$:

$$\Phi\left(m(t)\right) = 1 - \psi_1 + (\psi_1 - \psi_0) \frac{\zeta}{m(t) + \zeta}, \tag{6}$$

with: $\Phi(0) = 1 - \psi_0$, $\lim_{x \longrightarrow \infty} \Phi(x) = 1 - \psi_1$, $0 < \psi_0 < \psi_1 < 1$, $\Phi'(x) < 0$ and $\Phi''(x) > 0$; ψ_1, ψ_0 and ζ are positive productivity parameters to be fixed. As AIDS-related expenditures rise, function $\Phi(.)$ goes down, which induces a lower productivity gap due to morbidity. In our numerical experiments, we fix $\psi_1 = 1$, $\psi_0 = 0.5$, and we let ζ vary from 1 to 10, with $\zeta = 5$ in the reference simulation, as in Corrigan et al. (2005).

4. Concerning the saving behavior, we rely on the previous empirical work of Freire (2002) on the South African case. Freire's work is based on the seminal model of Blanchard (1985), and as such, it is much likely to capture, among others, the effects of increasing mortality on saving decisions than the very arbitrary computation rule adopted by Cuddington and Hancock, which was so toughly criticized by Bloom and Mahal (1997b). Actually, our treatment is intermediate between the "pessimistic" modelling adopted by Cuddington and Hancock, according to whom all AIDS-related health expenditures are fully detrimental to accumulation, and the excessively "neutral" modelling of Young (2005), who postulates a constant saving rate in a Solow-like setting.

3.3 Estimation and forecasting strategy

In this section, we present the forecasting methodology and the projection of HIV prevalence. The forecasting needed first the estimation of the parameters of an assumed econometric specification underlying the data generating process. For this purpose, we retain a Gaussian $ARMA(p,q)$ process for which we describe below the estimation and the forecasting procedure. Further details on these statistical methods can be found in Gouriéroux and Monfort (1990) and Hamilton (1994).

3.3.1 Conditional likelihood estimation

A Gaussian $ARMA(p,q)$ process is described as

$$\begin{aligned} Y_t = \alpha &+ \phi_1 Y_{t-1} + \phi_2 Y_{t-2} + \cdots + \phi_p Y_{t-p} + u_t \\ &+ \theta_1 u_{t-1} + \theta_2 u_{t-2} + \cdots + \theta_q u_{t-q} \qquad t = 1, \cdots, T \end{aligned} \tag{7}$$

where $u_t \sim$ i.i.d $N(0,\sigma^2)$, and where the vector of population parameters $\boldsymbol{\theta} = (\alpha, \phi_1, \phi_2, \cdots, \phi_p, \theta_1, \theta_2, \cdots, \theta_q, \sigma^2)'$ is to be estimated. The approximation to the likelihood function is conditioned on both initial values of the $y's$ and $u's$. Assuming that the initial values for $\boldsymbol{y}_0 \equiv (y_0, y_{-1}, \cdots, y_{-p+1})'$ and $\boldsymbol{u}_0 \equiv (u_0, u_{-1}, \cdots, u_{-p+1})'$ are given, the sequence $\{u_1, u_2, \cdots, u_T\}$ can be computed from $\{y_1, y_2, \cdots, y_T\}$ by iterating on

$$\begin{aligned} u_t = y_t &- \alpha - \phi_1 y_{t-1} - \phi_2 y_{t-2} - \cdots - \phi_p y_{t-p} \\ &- \theta_1 u_{t-1} - \theta_2 u_{t-2} - \cdots - \theta_q u_{t-q} \qquad t = 1, \cdots, T \end{aligned} \tag{8}$$

The conditional log likelihood is given by

$$\begin{aligned} L(\boldsymbol{\theta}) &= \ln f_{Y_T, Y_{T-1}, \cdots, Y_1 | \boldsymbol{Y}_0, \boldsymbol{u}_0}(y_T, y_{T-1}, \cdots, y_1 | \boldsymbol{y}_0, \boldsymbol{u}_0; \boldsymbol{\theta}) \\ &= -\frac{T}{2}\ln(2\pi) - \frac{T}{2}\ln(\sigma^2) - \sum_{t=1}^{T} \frac{u_t^2}{2\sigma^2} \end{aligned} \tag{9}$$

In maximizing this log likelihood, we set the initial $y's$ and $u's$ to their expected values. That is $y_s = \alpha/(1 - \phi_1 - \phi_2 - \cdots - \phi_p)$ for $s = 0, -1, \cdots, -p+1$, and $u_s = 0$ for

$s = 0, -1, \cdots, -p+1$. Then, we proceed with iteration in (8) for $t = 1, \cdots, T$. We estimate a multiple $ARMA(p,q)$ model with $p = 2$ and $q = 2$ which turns out to be an estimate of six models. We finally select the model that optimizes the Schwarz Criterion. The selected model is used for forecasting purpose.

3.3.2 Forecasting

Now, consider forecasting the stationary and invertible $ARMA(p,q)$:

$$(1 - \phi_1 L - \phi_2 L^2 - \cdots - \phi_p L^p)(Y_t - \mu) = (1 + \theta_1 L + \theta_2 L^2 + \cdots + \theta_q L^q) u_t \tag{10}$$

where L is the lag operator and μ is the unconditional mean $\mathbb{E}(Y_t)$. The one-period-ahead forecast ($s = 1$) is given by

$$\begin{aligned}(\hat{Y}_{t+1|t} - \mu) = \;& \phi_1 \;\; (Y_t - \mu) + \phi_2(Y_{t-1} - \mu) + \cdots \\ +\;& \phi_p(Y_{t-p+1} - \mu) + \theta_1 \hat{u}_t + \theta_2 \hat{u}_{t-1} + \cdots + \theta_q \hat{u}_{t-q+1}\end{aligned} \tag{11}$$

with $\hat{u}$ generated recursively from $\hat{u} = Y_t - \hat{Y}_{t|t-1}$. Finally the s-period-ahead forecasts based on the *Wiener-Kolmogorov prediction formula* is

$$(\hat{Y}_{t+s|t} - \mu) = \begin{cases} \phi_1(\hat{Y}_{t+s-1|t} - \mu) + \cdots + \phi_p(\hat{Y}_{t+s-p|t} - \mu) + \theta_s \hat{u}_t + \cdots + \theta_q \hat{u}_{t+s-q} \\ \hfill s = 1, \cdots, q \\ \phi_1(\hat{Y}_{t+s-1|t} - \mu) + \cdots + \phi_p(\hat{Y}_{t+s-p|t} - \mu) \\ \hfill s = q+1, \cdots \end{cases}$$

where $\hat{Y}_{\tau|t} = Y_\tau$ for $\tau \leq t$.

3.3.3 Projection of HIV prevalence

To obtain the projected prevalence, we fit a double logistic curve of the form

$$p(t) = \left[\frac{e^{\alpha(t-\tau)}}{1 + e^{\alpha(t-\tau)}}\right] \left[\frac{a e^{-\beta(t-\tau)}}{1 + e^{\beta(t-\tau)}} + b\right] \tag{12}$$

where α is the rate of increase at the start of the epidemic, a denotes the peak value, β is the rate of convergence, b is the final prevalence level and τ shifts the whole curve backward. The value of α is chosen so that the doubling time is 1.5 years. The latter is chosen so that the doubling time at the beginning of the epidemic can be $\ln(2)/\alpha$. This means that $\alpha = \ln(2)/1.5$. As a result, for a given β, we have to find (numerically) the parameters a, b and τ solution of the non linear system

$$\mathcal{S}(a,b,\tau) = \begin{cases} p(0) = 0.1916 = \frac{e^{-\alpha\tau}}{1+e^{-\alpha\tau}} \left[\frac{a e^{\beta\tau}}{1+e^{\beta\tau}} + b\right] \\ p(2015) = 0.194 = \frac{e^{\alpha(15-\tau)}}{1+e^{\alpha(15-\tau)}} \left[\frac{a e^{-\beta(15-\tau)}}{1+e^{-\beta(15-\tau)}} + b\right] \\ \dot{p}(t) = 0 \iff \frac{\alpha}{e^{\alpha(t-\tau)}} \left[\frac{a e^{-\beta(t-\tau)}}{1+e^{-\beta(t-\tau)}} + b\right] = \frac{a\beta e^{-\beta(t-\tau)}}{\left[1+e^{-\beta(t-\tau)}\right]^2} \quad \text{with } t = 3. \end{cases} \tag{13}$$

where $\dot{p}(t) = \frac{\mathrm{d}p(t)}{\mathrm{d}t}$. The first equation of the system corresponds to the starting period ($t = 0$ for year 2000 where the prevalence is 0.1916), the second denotes the ending period ($t = 16$ for year 2015 where the prevalence is 0.194) and the third equation expresses the peak of the epidemic. The value $t = 3$ represents the difference between the peak year 2003 and the starting date 2000.

3.4 Findings

We now summarize the results of experiments performed, including the sensitivity analysis.

3.4.1 Selected forecasting results

Using the optimal forecasting procedure described above, we have generated several projections for the key variables of the model for time horizons approaching 2050.[8] In order to save space, we only comment on the most important of them, those which will definitely matter in the interpretation of our findings.

Insert Figures 1 to 5

Figures 1 to 5 depict respectively our estimation and forecasting for life expectancy at birth with AIDS for female and male, for saving without and with AIDS and for GDP without AIDS. Figure 6 is the forecasting of economically active population for females with AIDS. The figures display both median forecast and the density estimate of the forecasted values. For these series, the Schwarz Criterion is optimized by and $ARMA(2,0)$ for life expectancy at birth, and an $ARMA(1,1)$ both for saving and GDP.[9] We can observe that the forecasts for life expectancy at birth with AIDS for female (Figure 1) and male (Figure 2) display nonlinear patterns. However, there is clear decreasing trend for both over the whole horizon, 1990 to 2050. The declining trend is indeed sizeable, and could not be omitted in any serious long-run analysis of AIDS. The forecast for saving without (Figure 3) and with AIDS (Figure 4) are very stable over time, after 2020. The confidence intervals are rather excellent for both. We shall notably observe here that the gap in saving rates between the AIDS and non-AIDS cases does not deepen tragically after 2020. It is about 2% in 2015 and it is in average around 3% after 2020. It is sizeable drop but our forecasts do not deliver any dramatic fall in savings, induced by AIDS, that one can *a priori* fear given the multiple mechanisms undermining accumulation in such a situation. Finally note that GDP forecasts with or without AIDS (like in Figure 5

[8] In some cases, we use the procedure to extend previous forecasts available for shorter horizons to 2050. This is the case of saving rate forecasts, see Figures 3 and 4 below: Freire's forecasts extend to 2015; we take as given and then we use the optimal forecasting procedure to go beyond 2015.

[9] For example, the forecast of GDP at 2000 is used to compute the initial capital in the case without AIDS.

for the non-AIDS case) show a non-ambiguous increasing tendency. In particular, AIDS epidemics, while massive, is not likely to induce negative growth over long periods of time.

Insert Figures 6 to 8

We turn now to mortality and prevalence rates. Mortality shows up clearly in the declining patterns of the active population. Figure 6 is an example, it displays the forecasting of economically active population for females with AIDS. More clearly than any other forecast so far, the figure shows the dreadful long-run demographic impact of AIDS, a marked decline especially after 2020.

Figure 7 is a plot of HIV prevalence rate from 1980 to 2015 as constructed by the US Census Bureau. In order to obtain HIV prevalence projections, we first solve numerically[10] the non linear system given by $\mathcal{S}(a, b, \tau)$ for a, b and τ. We obtain $a = 0.4045$, $b = 0,0003$ and $\tau = -9.356$. These values are then plugged into relation (12) to determine HIV prevalence from 2016 to 2050. The result is provided in Figure 8. The scenario studied is therefore a continuous (but not very fast) decrease in this rate after 2015, which seems reasonable.

3.4.2 The economic growth of AIDS quantified

The most salient ingredients of the model are demographic. As explained just above, the heavy trends in mortality and life expectancy induced by AIDS will be definitely more dreadful after 2020. For example, the forecasted series of life expectancy at birth reach their trough around 2009, with about 44 years for both sexes, while the starting values are about 60 years. This aspect and other relevant demographic conditions are likely to induce a delayed effect of AIDS on economic growth. The channels are the size of active population in the medium-run (say between 2020 and 2040), and because we assume by (5) that life expectancy at birth is a determinant of individuals productivity at their working age, we are likely to get a delayed impact of AIDS on productivity, at least when the sensitivity of productivity to life expectancy (that is parameter β) is large enough. Actually the delayed effect turns out to be strong in all our simulations, as it transpires from the following figures for growth per capita when β varies from 0 to 1.[11]

Insert Figure 9 to 10

One can notice the following. First of all, whatever the value of β, the gap between the AIDS and no-AIDS scenarios is rather stable between 2010 and 2020, but then it gets sharply larger between 2020 and 2030. The gaps keeps increasing but at an apparently much lower pace between 2030 and 2040. Finally, the gap seems to be stable (or

[10]Implemented using GAUSS 6 non linear system routine.

[11]The growth rates reported in the figures are "cumulative" growth rates over the successive decades. In order to get an average annual growth rate over a given decade, on has to divide the associated growth rate registered during the decade by 10.

shrinks very slightly) after 2040. One can therefore extract at least three main qualitative conclusions from the simulations.

1. The demographic impact of AIDS has a clear delayed effect on economic growth, according to our simulations.

2. The most "dangerous" period for economic growth is the decade $2020-2030$, where the gap between the AIDS and the non-AIDS scenarios gets deepened.

3. Though the gap does not increase so sharply after 2030, all our simulation feature a long run economic growth effect of AIDS.

Let us move now to a more quantitative assessment. To start with a benchmark case, let us consider a "linear" Ben-Porath case, that is $\beta = 1$. Table 1 gives the results for different values of the health expenditures productivity parameter ζ.

Insert Table 1

Consider the case $\zeta = 5$ as in the benchmark considered by Corrigan et al. (2005). One can see that the gap in growth of GDP per capita does not move significantly between 2010 and 2020. From 2020 to 2030, the gap sharply moves from 0.89% to 2.78%, which translated in annual rates means that AIDS causes GDP per capita to decline by 0.19% in the period $2020-2030$, while such a decline is around 0.09% in 2000-2010 for example. Accordingly, the economic growth impact of AIDS in $2020-2030$ more than doubles its counterpart value over the decade $2000-2010$. The gap keeps increasing from 2030 to 2040, it reaches 3.18% in 2040 but at a lower pace. Finally, the gap shrinks to 2.84% at 2050 mainly due to HIV-AIDS prevalence rates' values at this horizon.
Our figures for the growth gaps are a bit lower than those put forward by Over (1992) for the ten most affected sub-Saharan Africa countries for the period 1990-2025. It is not fair to discuss here who has the most accurate estimate since all the set-ups developed to this end, including ours, have their own shortcomings and limitations. Most studies, like Over's and ours, point at sizeable effects. In our case, the growth gap can be close to 0.2% for yearly growth rate of GDP per capita, which is considerable. Our distinctive contribution is in the timing of the growth effect after the incorporation of the most likely medium and long-run demographic trends induced by AIDS (in the econometric sense of the expression "more likely"). According to our forecasts, the largest part of AIDS negative impact on economic growth is likely to take place between 2020 and 2030. Another striking finding is that the growth differential between the two scenarios tend to be almost stable from 2030 to 2050 featuring a kind of long-run effect of AIDS.

Incidentally our exercise shows that there might not be any conflict between the results obtained by authors like Bloom and Mahal (1997b), and others like Corrigan, Glomm and Mendez (2005) or McDonald and Roberts (2005): A relatively short term assessment *à la* Bloom and Mahal might not reveal any dramatic AIDS growth effect, while a medium/long term perspective, relying on the evolution of the determinants of human capital accumulation, might deliver the opposite message. It seems reasonable to think that the observed sharply declining pattern of life expectancy must have delayed effects, and the governments should act from now on to alleviate the expected effects of such a trend.

3.4.3 Sensitivity analysis

We conduct two kinds of sensitivity analysis. First, we allow the coefficient β to vary taking the values 0, 1/7, 1/5, 1/2 and 1. The second sensitivity analysis concerns function $\Phi(m(t))$ (relation 6). We allow ζ to vary from 1 to 10 for each value of β. The results are given in the tables below.

Insert Tables 2 to 6

A first conclusion should be that the variation of parameter ζ of the health expenditures function does not change generally the first two decimals of the obtained growth per capita gap figures. The same type of findings is reported by Corrigan et al. (2005). Much more importantly, and not surprisingly, the gap figures increase with the sensitivity parameter β. A comparison between the polar cases $\beta = 0$ and $\beta = 1$ is useful to dig deeper in the findings. In the first case (and $\zeta = 5$), the gap jumps from 0.65 percentage point in 2020 to 2.65 percentage points in 2030, rises slightly to 2.87 in 2040, before coming back to the 2030 level in 2050. When $\beta = 1$, the size of the jump between 2020 and 2030 is naturally bigger, but the difference is even larger in 2040: the difference between the two gaps is about 0.13 in 2030, it is more than the double (equal to 0.31) in 2040. Therefore, while the fall in active population is probably, and by far, the main factor behind AIDS impact on economic growth during the decade 2020-2030, the Ben-Porath mechanism seems more relevant in the posterior decade. Both demographic factors are consequently key to understand the medium and long-run economic growth effects of AIDS in our scenarios, contrary to physical capital accumulation which does not seem to be the main story for these time horizons.

4 Conclusion

In this paper, we have first reviewed the main aspects of the empirical debate on the economic growth impact of epidemics. More specifically, we have focused on the debate on AIDS/HIV impact. We have shown how this debate is actually extremely disputed. A view however emerges according to which the loss in human capital might be truly devastating for long-run growth. We develop a simple general equilibrium model *á la* Solow extending previous work by Cuddington and Hancock. In particular, the sharply declining life expectancy patterns are clearly reflected in the enlarged model in the production function through a generic Ben-Porath mechanism (shorter lives imply shorter schooling times and therefore lower labor productivity). AIDS-related health expenditures are incorporated as well. Applied to the South African case, the model enhances the following aspect: while a relatively short term assessment might not reveal any dramatic AIDS growth effect, the medium/long run impact can be truly sizeable.

Our main contribution is to put forward a reasonable timing for the economic growth impact of AIDS under some widely accepted demographic scenarios. We first point out that the demographic impact of AIDS has a clear delayed effect on economic growth. Next, we show that the most "dangerous" period for economic growth is the decade $2020-2030$, where the gap between the AIDS and the non-AIDS scenarios gets deepened. Finally, Though the gap does not increase so sharply after 2030, all our simulation feature a long run economic growth effect of AIDS.

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Table 1: Growth rates (in %) of GDP per capita: gap between the AIDS and no-AIDS scenarios for varying the health productivity parameter ζ for $\beta = 1$

ζ	1	2.5	5	10
Year	GDP per cap	GDP per cap	GDP per cap	GDP per cap
2010	0.86	0.86	0.87	0.89
2020	0.89	0.89	0.89	0.89
2030	2.78	2.78	2.78	2.78
2040	3.18	3.18	3.18	3.18
2050	2.84	2.84	2.84	2.84

Table 2: Growth rates (in %) of GDP per capita: gap between the AIDS and no-AIDS scenarios for varying the health productivity parameter ζ for $\beta = 1/2$

ζ	1	2.5	5	10
Year	GDP per cap	GDP per cap	GDP per cap	GDP per cap
2010	0.85	0.85	0.86	0.89
2020	0.88	0.88	0.88	0.88
2030	2.72	2.72	2.72	2.72
2040	3.02	3.02	3.02	3.02
2050	2.74	2.74	2.74	2.74

Table 3: Growth rates (in %) of GDP per capita: gap between the AIDS and no-AIDS scenarios for varying the health productivity parameter ζ for $\beta = 1/5$

ζ	1	2.5	5	10
Year	GDP per cap	GDP per cap	GDP per cap	GDP per cap
2010	0.84	0.84	0.85	0.89
2020	0.87	0.87	0.87	0.87
2030	2.68	2.69	2.68	2.68
2040	2.93	2.93	2.93	2.93
2050	2.68	2.69	2.69	2.68

Table 4: Growth rates (in %) of GDP per capita: gap between the AIDS and no-AIDS scenarios for varying the health productivity parameter ζ for $\beta = 1/7$

ζ	1	2.5	5	10
Year	GDP per cap	GDP per cap	GDP per cap	GDP per cap
2010	0.84	0.84	0.85	0.88
2020	0.86	0.86	0.86	0.86
2030	2.68	2.67	2.68	2.67
2040	2.91	2.92	2.92	2.90
2050	2.68	2.68	2.67	2.67

Table 5: Growth rates of both GDP and GDP per capita: gap between the AIDS and no-AIDS scenarios for varying the health productivity parameter ζ for $\beta = 1/10$

ζ	1	2.5	5	10
Year	GDP per cap	GDP per cap	GDP per cap	GDP per cap
2010	0.83	0.84	0.85	0.88
2020	0.86	0.86	0.86	0.86
2030	2.67	2.67	2.67	2.66
2040	2.90	2.90	2.90	2.90
2050	2.67	2.67	2.67	2.66

Table 6: Growth rates (in %) of GDP per capita: gap between the AIDS and no-AIDS scenarios for varying the health productivity parameter ζ for $\beta = 0$

ζ	1	2.5	5	10
Year	GDP per cap	GDP per cap	GDP per cap	GDP per cap
2010	0.83	0.84	0.85	0.87
2020	0.86	0.86	0.86	0.86
2030	2.65	2.65	2.65	2.65
2040	2.87	2.87	2.87	2.87
2050	2.65	2.65	2.65	2.65

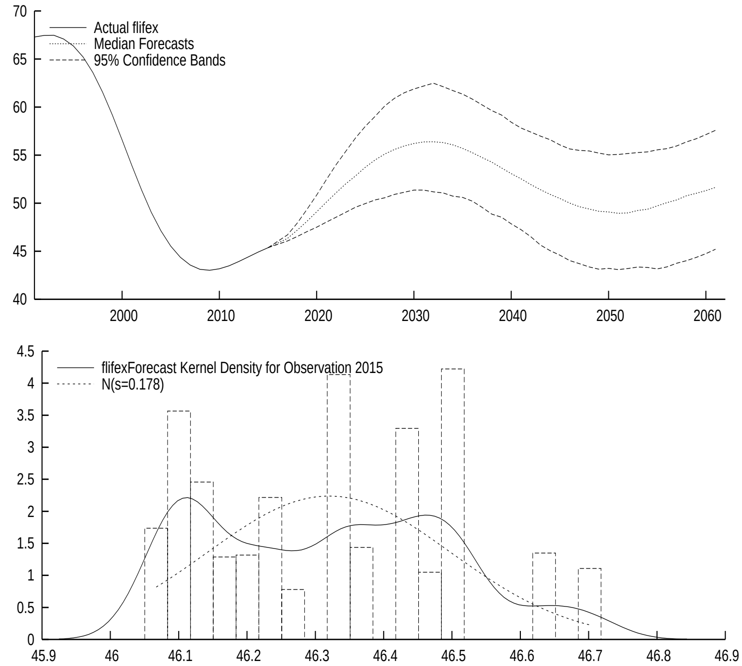

Figure 1: Forecasting of life expectancy at birth, female

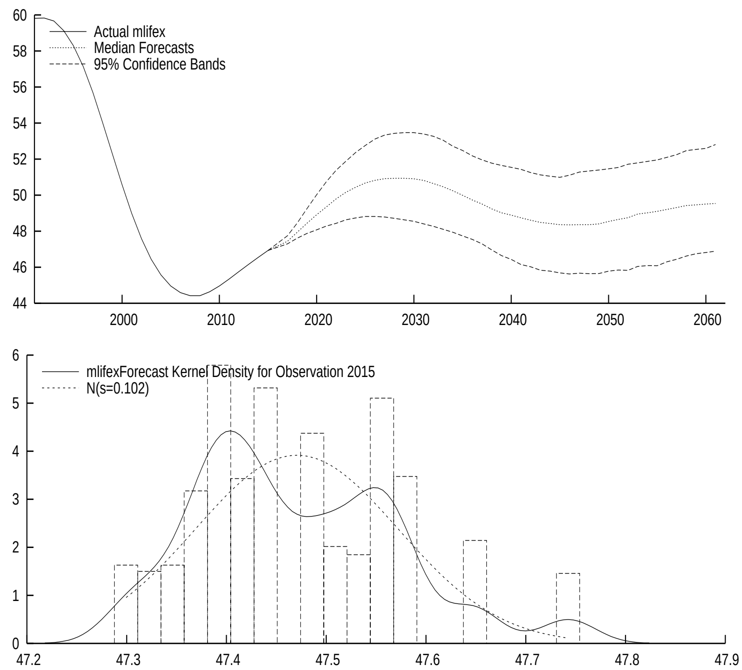

Figure 2: Forecasting of life expectancy at birth, male

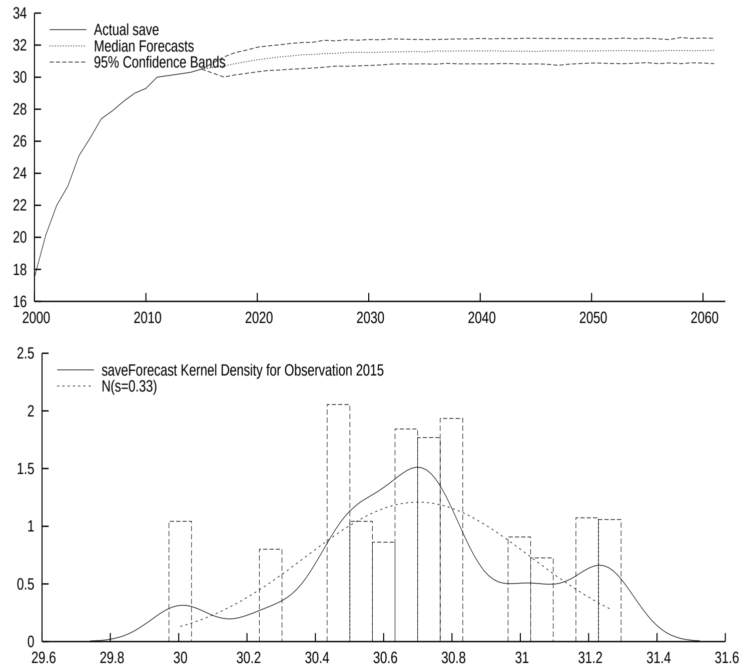

Figure 3: Forecasting of saving without AIDS based on Freire (2002)

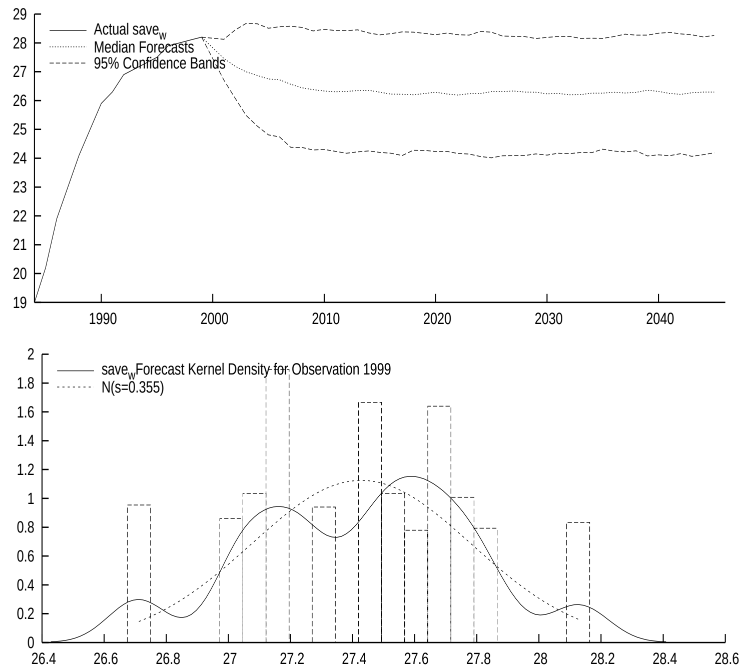

Figure 4: Forecasting of saving with AIDS based on Freire (2002)

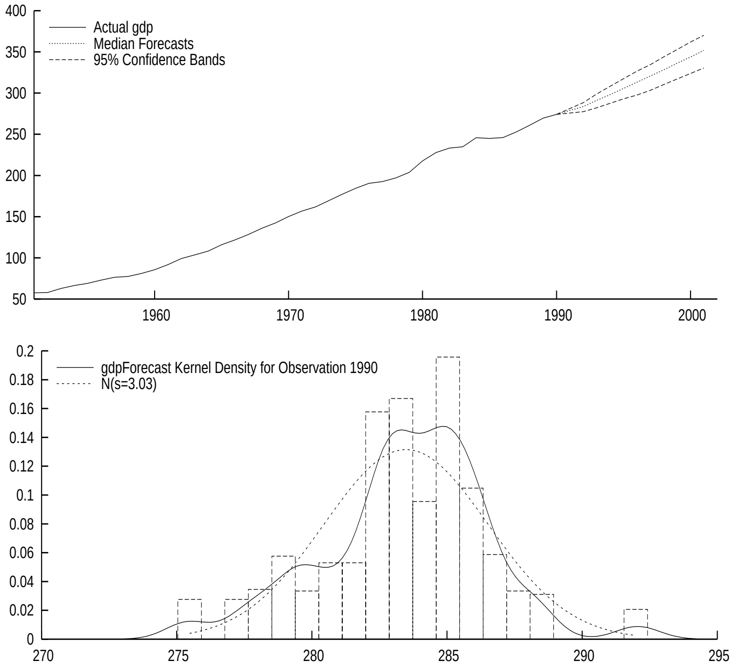

Figure 5: Forecasting of GDP without AIDS

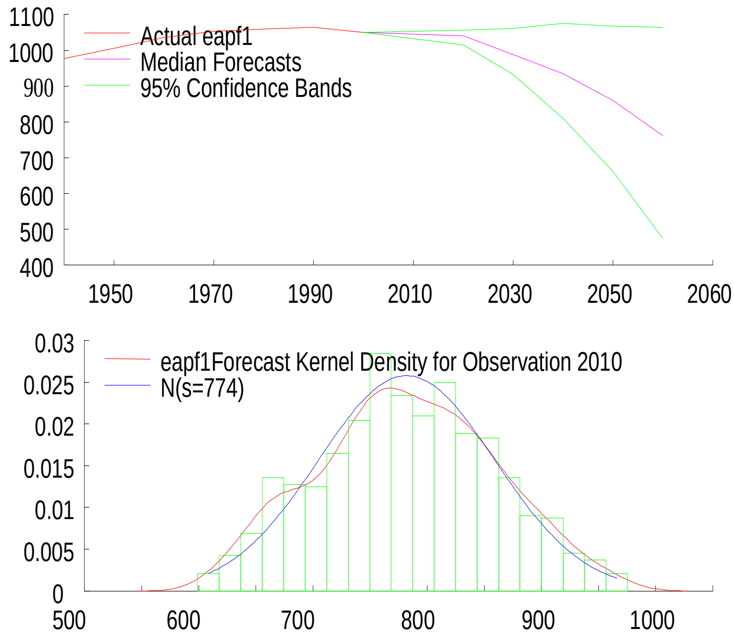

Figure 6: Forecasting of economically active population with AIDS, female

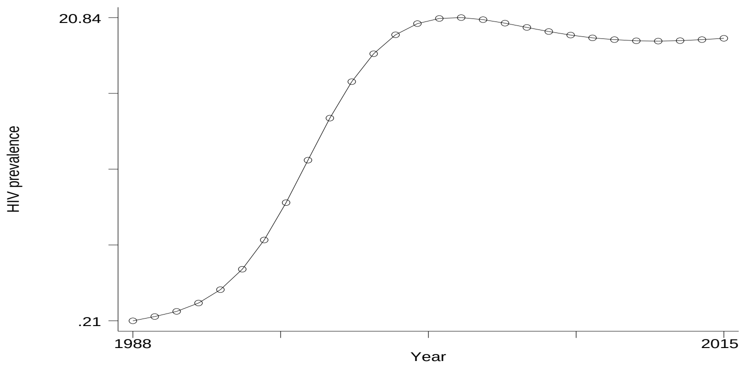

Figure 7: HIV prevalence in percent 1988-2015

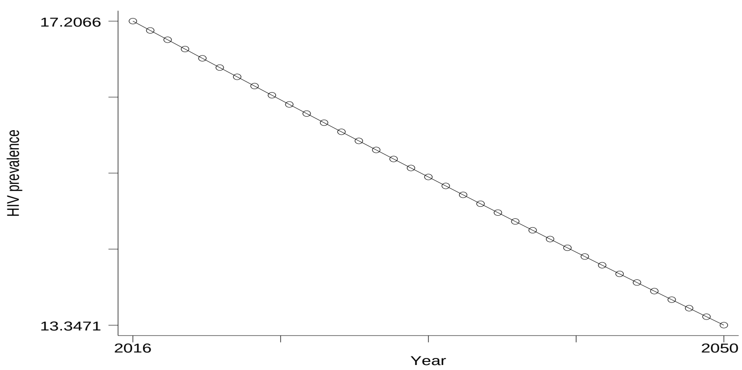

Figure 8: HIV prevalence 2016-2050

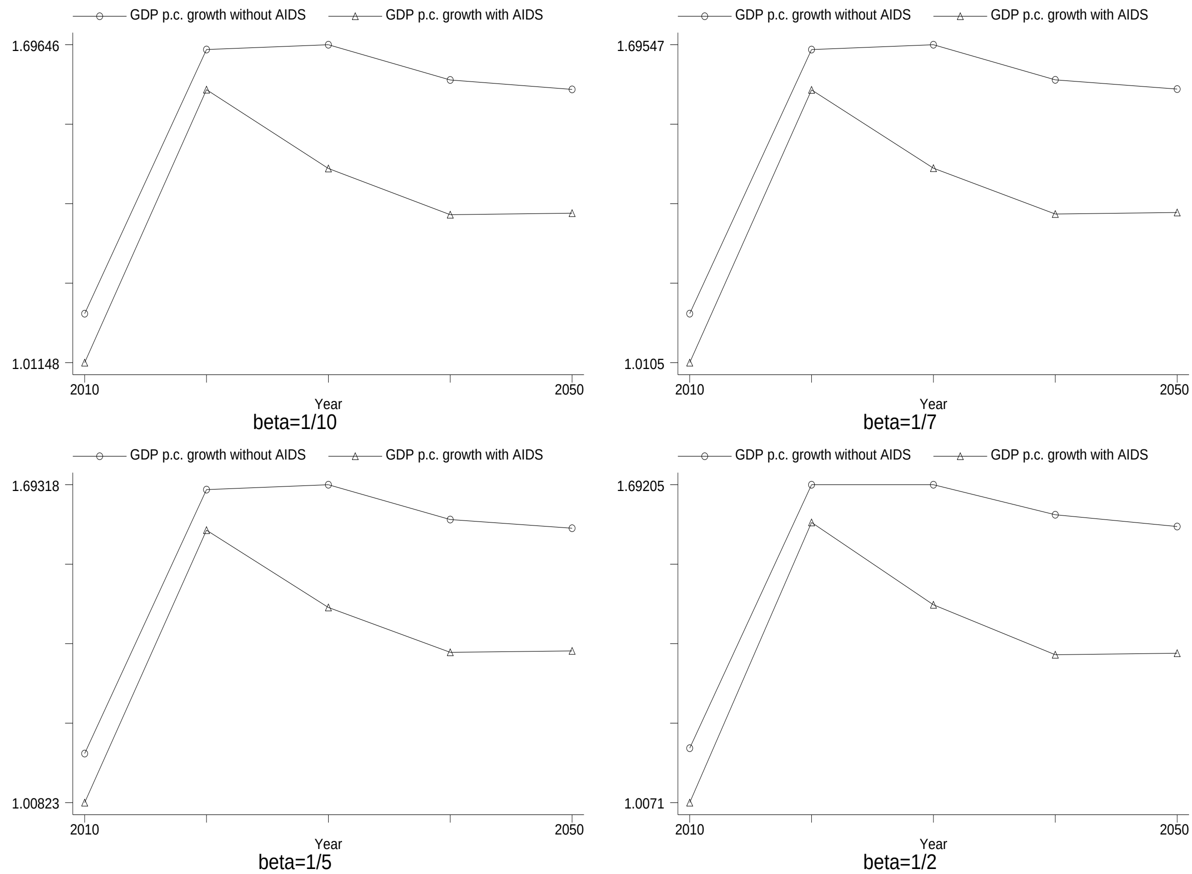

Figure 9: GDP growth rate per capita with and without AIDS

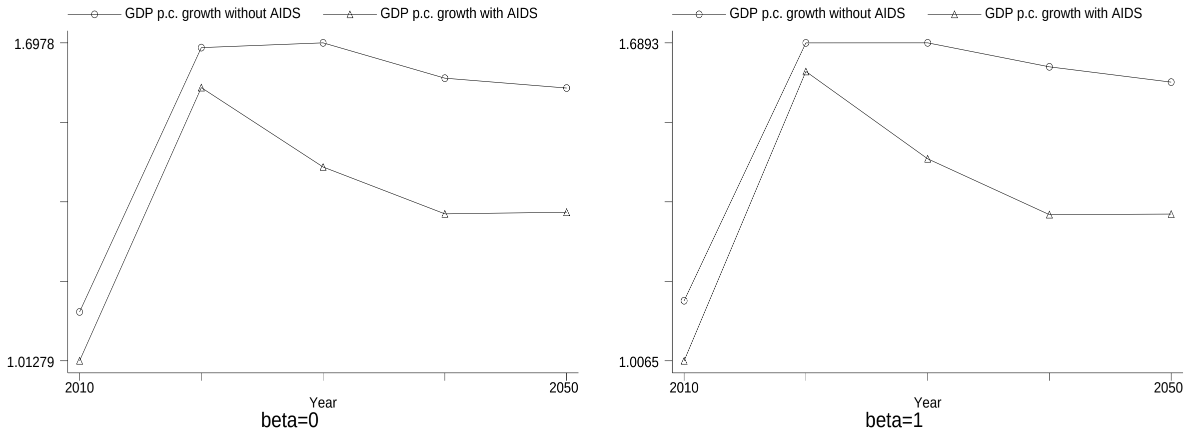

Figure 10: GDP growth rate per capita with and without AIDS: polar cases ($\beta = 0$ and $\beta = 1$)

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